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Comparison of the prognostic role of Q waves and inverted T waves in the presenting ECG of STEMI patients

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Abstract

Background: Both Q waves and T-wave inversion (TWI) in the presenting ECG are associated with a progressed stage of myocardial infarction, possibly with less potential for myocardial salvage with reperfusion therapy. Combining the diagnostic information from the Q- and T-wave analyses could improve the prognostic work-up in ST-elevation myocardial infarction (STEMI) patients.

Methods: We sought to determine the prognostic impact of Q waves and TWI in the admission ECG on patient outcome in STEMI. We formed four groups according to the presence of Q waves and/or TWI (Q+TWI+; Q−TWI+; Q+TWI−; Q−TWI−). We studied 627 all-comers with STEMI derived from two patient cohorts.

Results: The patients with Q+TWI+ had the highest and those with Q−TWI− the lowest 30-day and one-year mortality. One-year mortality was similar between Q−TWI+ and Q+TWI−. The survival analysis showed higher early mortality in Q+TWI− but the higher late mortality in Q−TWI+ compensated for the difference at 1 year. The highest peak troponin level was found in the patients with Q+TWI−.

Conclusion: Q waves and TWI predict adverse outcome, especially if both ECG features are present. Q waves and TWI predict similar one-year mortality. Extending the ECG analysis in STEMI patients to include both Q waves and TWI improves risk stratification.

KEYWORDS
ECG, Q wave, STEMI, T-wave inversion

1 | INTRODUCTION

The risk of death varies among patients with ST-elevation myocardial infarction (STEMI). Specific risk scores have been developed to improve risk assessment. The TIMI (thrombolysis in myocardial infarction) risk score applies clinical data to assess the risk of death (Morrow et al., 2000). ECG-based risk scores were introduced to aid in the assessment of acuteness of the ischemic process (Wilkins et al., 1995) and the area at risk (Aldrich et al., 1988). However, the presenting ECG in STEMI provides prognostic tools without need for complex analyses. The Sclarovsky-Birnbaum grading of ischemia classifies STEMI patients into two categories according to the distortion of the terminal portion of the QRS complex: grade 3 ischemia predicts higher short- and mid-term mortality and a larger infarct with a higher thrombus burden and more microvascular damage (Birnbaum, Birnbaum, & Birnbaum, 2014). However, this score applies only to patients with positive T waves in the leads with ST elevation. Another "at-a-glance" classification of STEMI is simply based on the analysis of Q and T waves in the ECG at presentation. "Evolving myocardial infarction" (EMI) is defined as pathological Q waves and/or inverted T waves, while these changes are absent in the "Pre-infarction syndrome" (PIS) (Sclarovsky, 1999). In the DANAMI-2 trial, patients with PIS had more advantage of primary percutaneous coronary intervention (pPCI) than of fibrinolytic

therapy (FT), while in the patients presenting with the EMI pattern, there was no significant difference between the two therapies (Eskola et al., 2007). The prognostic role of Q waves and T-wave inversions (TWI) in acute MI has been separately investigated in several studies. Q waves are the hallmark of myocardial necrosis, but they may also represent reperfusion (Blumenthal, Wang, & Pang, 1975; Horan, Flowers, & Johnson, 1971; Savage, Wagner, Ideker, Podolsky, & Hackel, 1977). Q waves predicted nonpatency of the culprit artery (Wong et al., 1999), a larger infarct (Delewi et al., 2013; Raitt et al., 1995) and higher 30-day and one-year mortality (Birnbaum et al., 1997; Siha et al., 2012; Wong et al., 2006). Post-treatment TWI in STEMI was associated with culprit artery patency and a favorable outcome (Corbalan et al., 1999; Doevendans et al., 1995; Lee et al., 2017; Matetzky et al., 1994; Ophuis et al., 2000; Sgarbossa et al., 2000), possibly indicating myocardial reperfusion. However, inverted T waves in the presenting ECG have been associated both with patency (Hira, Moore, Huang, Wilson, & Birnbaum, 2014) and nonpatency (Wong et al., 1999) of the culprit artery. Combining the diagnostic information from the Q- and T-wave analyses could improve the prognostic work-up in STEMI patients. Accordingly, the aim of this study was to establish the prognostic role of Q waves and TWIs in a real-life STEMI population.

2 | METHODS

This study comprises two Finnish nonrandomized STEMI populations. The STEMI 2005 study was conducted in the region of the Tampere University Hospital with a population of ~1.2 M. Data on the incidence, demographics, treatment strategies and delays were collected for consecutive STEMI patients (n = 310) in four hospital districts during a six-month period (Nikus et al., 2008). Regarding reperfusion therapy, both pPCI and FT were used. The study was observational, and treatment choices were based on prevailing international and regional guidelines.

In the HUS-STEMI study, patients (n = 448) were included during one year (2007–2008) in the district of the Helsinki University Central Hospital (Viikila et al., 2013). The study was observational, and the choice of reperfusion therapy was based on the decision by a consulting cardiologist. FT was recommended for hemodynamically stable patients when the time from symptom onset to treatment was ≤3 hr. As in the STEMI 2005 study, use of ancillary anti-thrombotic therapy and other therapeutic decisions were based on prevailing guidelines.

There were no prespecified exclusion criteria in the two studies. The inclusion criteria were as follows:

Acute chest pain/discomfort and

1. ST-elevations of ≥0.2 mV in at least 2 of the leads V1–3 or
2. ST-elevations of ≥0.1 mV in at least 2 other contiguous leads (V4–6; I, aVL; II, III, aVF) or
3. New or presumably new left bundle branch block.

We used troponin T with a cutoff <0.01 μg/L. Renal insufficiency was defined as creatinine >150 μM (1.70 mg/dl) on admission.

A written informed consent was signed by the patients before enrolment. The local Ethics Committees approved the study protocol.

For the present study, mortality data were collected from the official national registry (Statistics Finland), which records 100% of deaths of Finnish citizens at home and nearly 100% abroad. Regarding other endpoints, data from the two studies were used.

The primary endpoint was mortality at one year. Other prespecified endpoints were in-hospital mortality, 30-day mortality, and 30-day MACE (major adverse cardiovascular events: a composite of cardiovascular death, stroke, re-infarction and new, unplanned revascularization procedures).

The definition of "no revascularization therapy" (NRT) was: no FT or angiography within 4 hr from presentation.

The ECG data were analyzed by one investigator (KK), who at the time of the analysis was blinded to clinical data. In case of doubt, a mutual agreement was sought with two senior cardiologists (ME, KN). We found that in 46 patients the ST changes did not meet the established cutoff values, although included by the investigators on-site. These patients were excluded. Likewise, patients with a missing/incomplete (n = 25), or noninterpretable ECG (n = 8) were excluded. Patients with a wide QRS (>120 ms) were excluded as well (n = 52) (Figure 1).

Pathological Q waves were defined according to the Third Universal Definition of Myocardial Infarction (Thygesen et al., 2012). Any TWI ≥0.05 mV was considered as inverted. Q waves and TWIs outside the leads with maximum ST elevation were ignored. We formed four groups according to the presence of Q waves and/or TWIs (Q+TWI+, Q−TWI+, Q+TWI−, and Q−TWI−) (Figure 2).

The data were analyzed with SPSS Statistics 25. We compared the four groups with respect to different prespecified variables. In categorical variables, we used the χ^2 test or when applicable, Fisher's exact test. The distribution of all continuous variables was skewed, so we used median values and the independent-samples Mann–Whitney U test for the difference between the two groups. Interquartile ranges were defined using the weighted average. We performed a logistic regression univariate analysis using one-year mortality as the endpoint. We present odds ratios with 95% confidence intervals (CI). We selected the variables with a p value <0.1 to the multivariable analysis.

3 | RESULTS

The Q+TWI+ pattern was found in 29 patients (4.6%), Q−TWI+ in 50 patients (8.0%), Q+TWI− in 130 patients (20.7%) and Q−TWI− in 418 patients (66.7%) (Figure 1). The distribution of the four groups was similar in the STEMI 2005 and HUS-STEMI studies (p = 0.304).

The baseline characteristics of the four ECG categories are shown in Table 1. The Q+TWI+ group clearly had the highest proportion of males (82.8%), as the proportion in the other three groups was 44.0%–67.7% (p = 0.003). The proportion of patients with

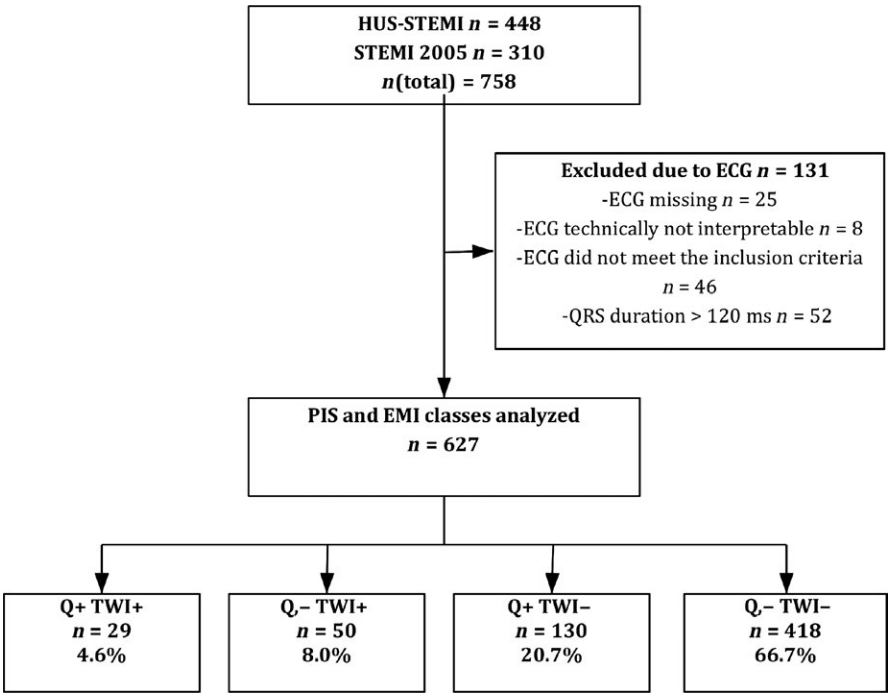

FIGURE 1 The number of included and excluded patients and the number of patients in the four ECG groups

diabetes was highest in Q+TWI+ (34.5%) and lowest in Q−TWI− (15.6%), $p = 0.033$. Killip class >1 was seen more often in the groups with Q waves (44.8% in Q+TWI+ and 39.5% in Q+TWI−) as compared to those with no Q waves (30.0% in Q−TWI+ and 23.8% in Q−TWI−), $p = 0.001$. Likewise the location of the STEMI more often was anterior in the groups with Q waves ($p < 0.001$) with the highest proportion in the Q+TWI− group (76.9%).

There was no significant difference between the groups in the rate of pPCI. However, FT more often was given to the patients with no TWI (55.4% in Q+TWI− and 58.4% in Q−TWI−) as compared to those with TWI (24.1% in Q+TWI+ and 28.0% in Q−TWI+), $p < 0.001$. Also, the proportion of patients who received no immediate reperfusion therapy differed between the groups being highest in Q+TWI+ (34.5%) and lowest in Q−TWI− (8.4%), $p < 0.001$. There was no significant difference in the use of medication between the groups.

Time from symptom onset to ECG was longest in the patients with TWI (365 min in Q+TWI+ and 184 min in Q−TWI+) and shortest in those with no TWI (89 min in Q+TWI− and 73 min in Q−TWI−), $p < 0.001$. The time from symptom onset to treatment was 498, 320, 153, and 142 min in the respective groups, $p < 0.001$.

Patient outcome according to the ECG categories is shown in Table 2. Thirty-day mortality was highest in patients with Q+TWI+, followed by Q+TWI−, Q−TWI−, and finally Q−TWI+ (20%, 14.4%, 6.8% and 6.4%, respectively, $p = 0.012$). One-year mortality was 31%, 19.2%, 9.8%, and 22% in the respective groups ($p < 0.001$). One-year mortality did not differ between the Q+TWI− and Q−TWI+ patients ($p = 0.677$, not shown in the tables).

The highest peak troponin levels were found in patients with Q+TWI− (3.23 μg/L), followed by Q+TWI+ (2.81 μg/L), Q−TWI+ (2.59 μg/L) and Q−TWI− (1.81 μg/L), p value for the difference was 0.002. In the Kaplan–Meier survival analysis, the patients with Q+TWI+ had the worst outcome early-on (Figure 3). Patients with Q+TWI− and those with Q−TWI+ had similar one-year survival. The survival curves indicate high early mortality in the former group while in the latter group late mortality was higher. p Value for the difference in survival between the four groups was <0.001 (Log-Rank).

The results of the logistic regression analyses are shown in Table 3. In the logistic regression univariate analysis, all other groups were associated with higher one-year mortality as compared to Q−TWI−. In the multivariable analysis, Q+TWI+ was independently associated with one-year mortality (OR 7.14, 95%CI 2.05–24.9, $p = 0.002$), while Q+TWI− was not (OR 1.46, 95%CI 0.614–3.54, $p = 0.385$), and Q−TWI+ had a tendency toward independent association (OR 3.13, 95%CI 0.900–10.9, $p = 0.073$). Other variables independently associated with one-year mortality were age, hyperlipidemia, and Killip class >1.

4 | DISCUSSION

Q waves and inverted T waves have been recognized as ECG markers of a progressed stage of myocardial infarction since the dawn of STEMI diagnostics. The prognosis of the combination of Q waves and/or TWI (evolving myocardial infarction, EMI) was studied in the DANAMI-2 trial, where this ECG pattern was an independent risk factor for the composite endpoint of mortality, clinical infarction, and disabling stroke (Eskola et al., 2007). The present study is the first one to compare the prognosis of Q waves and TWI.

In the present study, Q waves and TWI showed different prognostic features. The patients with both Q waves and TWI had the worst outcome. One-year mortality was similar in patients with either Q+TWI− or Q−TWI+. Interestingly, the Kaplan–Meier analysis

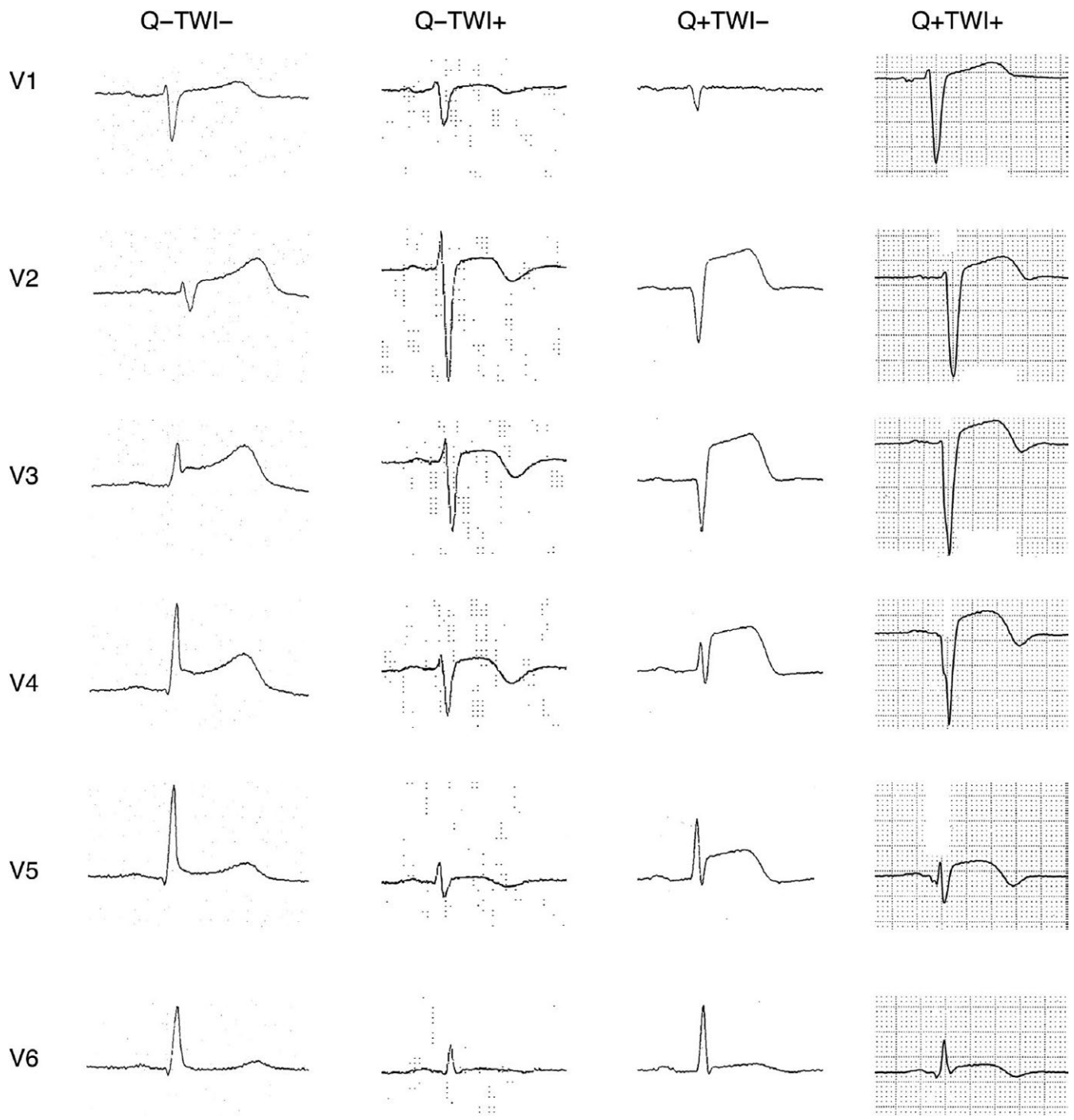

FIGURE 2 The four ECG groups showed in anterior STEMI (50 mm/s)

showed high early mortality in Q+TWI− and high late mortality in Q-TWI+. According to peak troponin values, patients with Q waves had larger infarcts, which could explain high early mortality. The relatively high late mortality of the Q−TWI+ group is more difficult to explain.

The immediate ECG changes caused by acute coronary artery occlusion are a positive, prominent T wave and ST elevation, features which qualify for the Q−TWI− classification (or preinfarction syndrome) (Nikus et al., 2010). Q waves and inverted T waves are mostly considered as later changes during the infarct process with less potential for myocardial salvage. However, our results imply that Q waves and TWI are different phenomena despite comparable outcome at one-year follow-up. Basically, TWI is seen in late-presenting STEMI patients, whereas Q waves imply a large infarct. Killip class was higher in patients with Q waves. As Q waves indicate more extensive myocardial damage, their association with higher Killip class is logical. Thus, a patient with both Q waves and TWI may often be a late-presenter with a large infarct. It is plausible that

TABLE 1 Baseline characteristics

	Q+TWI+ *n* = 29 *n* (%)	Q−TWI+ *n* = 50 *n* (%)	Q+TWI− *n* = 130 *n* (%)	Q−TWI− *n* = 418 *n* (%)	*p* Value
Gender (male)	24 (82.8)	22 (44.0)	88 (67.7)	273 (65.3)	0.003
Current smoker	14 (51.9)	13 (28.3)	44 (37.6)	148 (37.0)	0.254
Diabetes	10 (34.5)	10 (20.0)	29 (22.3)	65 (15.6)	0.033
Hyperlipidemia	11 (37.9)	15 (30.0)	64 (49.2)	188 (45.0)	0.113
Hypertension	16 (57.1)	22 (44.0)	79 (60.8)	223 (53.3)	0.206
Prior STEMI	5 (17.2)	3 (6.0)	18 (14.1)	35 (8.4)	0.093
Prior angina	10 (35.7)	14 (31.8)	30 (25.2)	115 (28.5)	0.663
Prior CHF	2 (7.1)	4 (8.0)	6 (4.6)	20 (4.8)	0.591
Prior TIA/stroke	2 (6.9)	4 (8.0)	7 (5.4)	32 (7.7)	0.846
Renal insufficiency	2 (6.9)	2 (4.0)	3 (2.3)	11 (2.6)	0.387
Prior PCI	1 (3.4)	5 (10.0)	9 (6.9)	22 (5.3)	0.461
Prior CABG	2 (6.9)	2 (4.0)	2 (1.5)	12 (2.9)	0.264
Killip class >1	13 (44.8)	15 (30.0)	51 (39.5)	99 (23.8)	0.001
STEMI in anterior location	16 (55.2)	23 (46.0)	100 (76.9)	150 (35.9)	<0.001
pPCI	12 (41.4)	24 (48.0)	41 (31.5)	139 (33.3)	0.143
FT	7 (24.1)	14 (28.0)	72 (55.4)	244 (58.4)	<0.001
NRT	10 (34.5)	12 (24.0)	17 (13.1)	35 (8.4)	<0.001
Aspirin	11 (37.9)	13 (26.0)	45 (34.6)	105 (25.2)	0.112
Clopidogrel	1 (3.4)	1 (2.0)	2 (1.5)	3 (0.7)	0.188
Warfarin	0 (0.0)	3 (6.0)	5 (3.8)	23 (5.5)	0.625
β-blocker	7 (24.1)	19 (38.0)	45 (34.9)	122 (29.3)	0.356
Calcium channel blocker	3 (10.3)	11 (22.0)	22 (16.9)	66 (15.8)	0.578
Statin	9 (31.0)	7 (14.0)	27 (20.8)	79 (18.9)	0.301
ACEi/ARB	11 (37.9)	15 (30.0)	35 (26.9)	93 (22.4)	0.172
	Median (quartiles)	**Median (quartiles)**	**Median (quartiles)**	**Median (quartiles)**	
Age (years)	65 (54–72)	68 (58–77)	65 (55–78)	66 (57–76)	0.831
Time from symptom onset to ECG (min)	356 (80–730)	184 (72–465)	89 (45–208)	73 (40–161)	<0.001
Time from symptom onset to treatment (min)	498 (285–940)	320 (235–795)	153 (94–299)	142 (85–240)	<0.001

Note. ACEi: angiotensin-converting-enzyme inhibitor; ARB: angiotensin receptor blocker; CABG: coronary artery bypass graft; CHF: congestive heart failure; FT: fibrinolytic therapy; NRT: no reperfusion therapy; PCI: percutaneous coronary intervention; pPCI: primary PCI; STEMI: ST-elevation myocardial infarction; TIA: transient ischemic attack.

this combination represents the STEMI category with the worst outcome in our classification.

In the present study, the proportion of anterior STEMI differed between the four ECG categories being highest in the patients with Q waves. Both Q waves (Wong et al., 2006) and inverted T waves (Hira et al., 2014) have been reported more often in the anterior leads. A high proportion of anterior STEMI in the EMI group was also reported in the DANAMI-2 trial (Eskola et al., 2007). There is no evidence for myocardial necrosis to develop faster in the anterior wall. We think that the criteria are more sensitive for anterior Q waves, and this also is possibly true for TWI. There may also be other differences in the typical evolving ECG patterns in STEMI of different locations.

In individual patients, myocardial necrosis progresses at different rates. Collateral flow and ischemic preconditioning are known to alleviate the ischemic damage (Ottani et al., 1995). Hence, the time from symptom onset may not be the best way to assess the evolution of the infarct process; ECG scoring systems, such as the Anderson-Wilkins score, may provide more accurate information (Sejersten et al., 2007).

The pathophysiologic role of Q waves in STEMI is controversial. In canine experiments, Q waves appeared only after the release of the coronary occlusion and seemed to indicate both myocardial reperfusion and damage (Blumenthal et al., 1975; Bodenheimer, Banka, Levites, & Helfant, 1976). Wong et al found Q waves to be

TABLE 2 Outcome with respect to Q waves and T-wave inversion (for abbreviations, see Table 1)

	All *n* (%) *n* = 627	Q+TWI+ *n* (%) *n* = 29	Q−TWI+ *n* (%) *n* = 50	Q+TWI− *n* (%) *n* = 130	Q−TWI− *n* (%) *n* = 418	*p* Value
Thirty-day follow-up						
Thirty-day CV mortality	47 (7.9)	4 (16.0)	3 (6.4)	14 (11.2)	26 (6.5)	0.126
Thirty-day AMI	29 (4.9)	2 (8.0)	2 (4.3)	7 (5.6)	18 (4.5)	0.705
Thirty-day stroke	11 (1.8)	0 (0)	0 (0)	4 (3.2)	7 (1.8)	0.655
Thirty-day new nonelective CABG/PCI	15 (2.5)	0 (0)	0 (0)	2 (1.6)	13 (3.3)	0.578
Thirty-day MACE	90 (15.1)	6 (24.0)	4 (8.5)	25 (20.0)	55 (13.8)	0.108
Lost for follow-up	31	4	3	5	19	
In-hospital mortality	45 (7.2)	5 (17.2)	4 (8.0)	12 (9.2)	24 (5.7)	0.077
1-year mortality	86 (13.7)	9 (31.0)	11 (22.0)	25 (19.2)	41 (9.8)	<0.001
	All Median (quartiles)	**Q+TWI+ Median (quartiles)**	**Q−TWI+ Median (quartiles)**	**Q+TWI− Median (quartiles)**	**Q−TWI− Median (quartiles)**	***p* Value**
Maximum troponin μg/L	2.17 (0.588–5.38)	2.81 (1.31–4.59)	2.59 (0.750–4.78)	3.23 (0.930–8.80)	1.81 (0.425–4.67)	0.002

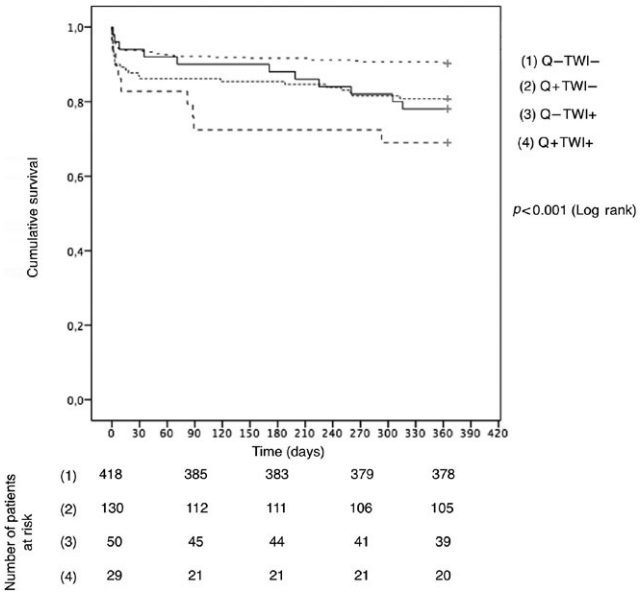

FIGURE 3 The Kaplan–Meier analysis showing the survival of patients with Q−TWI−, Q+TWI−, Q−TWI+, and Q+TWI+ in one-year follow-up

associated with nonpatency of the infarct-related artery (Wong et al., 1999). In the study of Raitt et al, abnormal Q waves were seen in 53% of the patients presenting within 1 hr from symptom onset, which makes their association with myocardial necrosis questionable, although Q waves did predict larger final infarct size (Raitt et al., 1995). Q waves may be associated with a larger area at risk. In cardiac magnetic resonance imaging, Q waves were a better indicator of infarct size than of infarct transmurality (Moon et al., 2004). Baseline Q waves have been shown to predict slower ST resolution even when the culprit artery is patent and the patients presents early, perhaps reflecting microvascular damage (Wong et al., 2002). Logically, higher mortality with Q waves has been reported in patients treated with FT (Wong et al., 2006) or primary PCI (Siha et al., 2012). Like previous studies, our study showed larger infarcts in the patients with Q waves.

Also the issue of inverted T waves in the ECG in STEMI is somewhat controversial. In patients presenting with ST elevations, development of negative T waves (or terminally inverted T waves) post-treatment is undoubtedly a predictor of favorable outcome, indicating culprit artery patency and reduced short-term mortality in patients treated with FT (Corbalan et al., 1999; Doevendans et al., 1995; Matetzky et al., 1994; Ophuis et al., 2000; Sgarbossa et al., 2000) or primary PCI (Lee et al., 2017). The role of TWI in the presenting ECG in STEMI is less obvious. In STEMI patients treated with pPCI, better myocardial recovery was reported with inverted T waves (Sorensen et al., 2009). In the HERO-1 study, inverted T waves in the presenting ECG were associated with nonpatency of the culprit artery (Wong et al., 1999). Contradictory, in the study by Hira et al., TWI in the presenting ECG predicted infarct-related artery patency with a median delay from symptom onset of 5 hr. Alsaab et al., (2014 also found an association between inverted T waves and culprit artery patency in a study with very short time delays: 50% of the patients with TWI had PCI within 1 hr from the symptom onset. In these studies, mortality was not assessed. In the study of Herz et al, TWI in the presenting ECG was a predictor of worse outcome in the late-presenting (>2 hr) patients, while in the early presenters, there was a tendency toward favorable outcome (Herz et al., 1999). Shimada, Po, Kanei, and Schweitzer (2013) found more in-hospital major adverse cardiac events, longer hospital stay and less ST resolution after PCI in patients with TWI. Although the mean delay to treatment was very long (28 hr) in

TABLE 3 Logistic regression univariate and multivariable analyses with one-year mortality as the endpoint

	Univariate analysis			Multivariable analysis		
	OR	95% CI	*p* Value	OR	95% CI	*p* value
ECG						
Q−TWI−	Ref.			Ref.		
Q+TWI−	2.19	1.27–3.77	0.005	1.46	0.614–3.54	0.385
Q−TWI+	2.59	1.23–5.45	0.012	3.13	0.900–10.9	0.073
Q+TWI+	4.14	1.77–9.68	0.001	7.14	2.05–24.9	0.002
Male	0.436	0.275–0.690	<0.001	1.72	0.782–3.765	0.179
Age	1.10	1.07–1.13	<0.001	1.06	1.02–1.10	0.001
Current smoker	0.495	0.265–0.924	0.027	1.07	0.442–2.59	0.881
Diabetes	2.65	1.59–4.40	<0.001	1.56	0.687–3.53	0.289
Hyperlipidemia	0.497	0.304–0.811	0.005	0.413	0.183–0.930	0.033
Hypertension	1.895	1.17–3.07	0.009	2.03	0.756–5.43	0.160
Prior STEMI	1.65	0.837–3.24	0.149			
Prior angina	1.94	1.14–3.29	0.014	1.24	0.603–2.54	0.561
Prior CHF	8.62	4.12–18.0	<0.001	1.43	0.402–5.07	0.582
Prior TIA/stroke	3.20	1.62–6.30	<0.001	1.66	0.549–5.00	0.370
Renal insufficiency	11.2	4.21–29.8	<0.001	1.79	0.316–10.1	0.512
Prior PCI	0.982	0.372–2.59	0.971			
Prior CABG	2.51	0.871–7.22	0.088	1.86	0.288–12.1	0.514
Killip class >1	10.8	6.39–18.3	<0.001	5.99	2.83–12.1	<0.001
Time from symptom onset to ECG	1.00	0.999–1.00	0.911			
Time from symptom onset to treatment	1.00	0.999–1.00	0.887			
STEMI in anterior location	1.41	0.894–2.22	0.14			
NRT	Ref.			Ref.		
FT	0.374	0.204–0.687	0.002	1.39	0.449–4.31	0.567
pPCI	0.353	0.182–0.684	0.002	1.25	0.397–3.92	0.705
Aspirin	2.35	1.47–3.76	<0.001	1.63	0.714–3.70	0.247
Clopidogrel	2.55	0.487–13.4	0.267			
Warfarin	2.31	0.998–5.35	0.050	1.24	0.284–5.44	0.772
β-blocker	3.24	2.02–5.18	<0.001	1.57	0.704–3.48	0.272
Calcium channel blocker	2.17	1.27–3.70	0.005	1.23	0.504–3.01	0.648
Statin	1.02	0.577–1.81	0.938			
ACEi/ARB	1.64	1.00–2.70	0.050	0.521	0.218–1.24	0.141

Note. For abbreviations, see Table 1.

the patients with TWI, the results were similar in the patients presenting early (<2 hr).

It seems that TWIs in the presenting ECG and those developing post-treatment have different outcome. In the presenting ECG, TWI indicates a later stage of the infarct with worse outcome—at least in the late-presenting patients. TWI post-treatment is a sign of reperfusion associated with favorable outcome. The present study parallels with former studies in showing higher mortality in STEMI patients with inverted T waves. In the present study, most of the TWI+ patients were late-presenters as defined in the study by Herz et al. (1999; median time from symptom onset to treatment 320–498 min in the TWI+ groups).

5 | LIMITATIONS

The study consisted of two study populations, which could result in nonuniformity. However, the design and the case report forms of the two studies were almost identical and the distribution of the studied ECG categories was similar in the two studies. Hence, these differences should not represent major obstacles regarding the interpretation of the study results.

At the time of the studies, some hospitals used troponin I instead of troponin T. Due to this fact, we had to exclude some (n = 49) patients from the analysis, which included troponin.

6 | CONCLUSION

Simply classifying STEMI patients into four categories based on Q- and T-wave analyses aids in outcome prediction. In all-comers, both Q waves and TWI predicted higher one-year mortality than ST elevations only. Q waves were associated with larger infarcts and TWIs with longer treatment delays. The patients with both Q waves and TWI had highest one-year mortality. Future studies in different patient populations and treatment strategies are required to better establish the role of this ECG classification in clinical routine practice.

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CONFLICTS OF INTEREST

The authors declare that there are no conflicts of interest.

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